EFFECT OF LOWER EXTREMITY STRENGTH TRAINING ON GAIT IN CEREBRAL PALSY CHILDREN

Project

Submitted to

The Tamilnadu Dr. MGR Medical University

In partial fulfillment for the degree of

MASTER OF PHYSIOTHERAPY

(**PEADIATRIC PHYSIOTHERAPY**)

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CERTIFICATE

The work embodied in the thesis entitled **"Effect of Lower Extremity Strength Training on Gait in Cerebral Palsy Children"** Submitted to **The Tamilnadu Dr. MGR Medical University** in partial fulfillment for the degree of Master of physiotherapy was carried out by candidate bearing register number **271540041** at Cherraan's College of Physiotherapy under my supervision. This is an original work done by her and has not been submitted in part or full for any other degree/diploma at this of any other university/ institute. The thesis is fit to be considered for evaluation for award of the degree of master of physiotherapy

Signature of supervisor — **Principal**

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INTERNAL EXAMINER — **EXTERNAL EXAMINER**

Project Work Evaluated On: ______________________.

DECLARATION BY THE STUDENT

The work embodied in the thesis entitled "**EFFECT OF LOWER EXTREMITY STRENGTH TRAINING ON GAIT IN CEREBRAL PALSY CHILDEREN**" Submitted to **The Tamilnadu Dr. MGR Medical University** in partial fulfillment for the degree of Master of physiotherapy was the original work carried out by me and has not been submitted in part or full for any other degree / diploma at this or any other university / institute. All the ideas and references have been duly acknowledged.

Signature of supervisor	**signature of student**
Mrs. Aroona MPT	Bavapriya .P
(professor)	

Date: ___________________

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CONTENTS

INTRODUCTION

Cerebral palsy is a collection of disorders characterized by an insult to the developing brain that produces physical disability as the primary or distinguishing feature **Dr.Little** described cerebral palsy as a persistent disorder of posture and movement appearing early in life and due to a developmental non progressive disorder of the brain.

The spastic form of cerebral palsy is the most common and in those patients, additional clinical signs may include, muscle shortening, diminished selective control and weakness. It is well recognized that muscle weakness is a major impairment in people with cerebral palsy. Motor problems affecting gait includes disruption to both neuromuscular and musculoskeletal system. In patients with neurological pathology musculoskeletal problem are secondary to primary neuromuscular problem.

Central lesions of the CNS results in impaired programming affecting leg muscle activation. Impaired programming can manifest in gait as.

- Inability to recruit a muscle appropriately.
- Increased activation of muscle that is unrelated to spasticity mediated stretch.
- Inability to modulate a muscle activity throughout the gait cycle.

Many children with cerebral palsy quickly became exhausted because of the disease’s impact on the muscle. According to the researchers the average speed of cerebral palsy kids is about 40m/s or 57% of normal walking speed. These children walk slowly they required high energy expenditure often lack endurance and suffer from muscle weakness.

While spasticity was once thought to be the primary contributor to the motor dysfunction noted in cerebral palsy, many have challenged this perspective and now consider ‘negative’ signs such as muscle weakness to be more harmful to function.

The lower level of physical activity observed in this population is one potential contributor to weakness[10] but is hardly the sole explanation. Other possible factors include decreased central input to the muscle due to a pyramidal tract insult[11], changes in elastic properties of the muscles themselves[12], aberrations in the reciprocal inhibition pathways in agonist-antagonist muscle pairs[13] and heightened stretch responses or spasticity.

(Patla 1995; Buschner and DeLateur 1991) Walking[21] does not normally tax the various lower extremity muscle groups to their full capacity. The only muscle that comes close to their maximum output during gait is the ankle Plantar flexor, which normally provide a major source of propulsive power. In the presence of plantar flexor weakness, alternative power source are used resulting in changes in locomotors characteristic like stride length & velocity.

Several researchers have shown a positive association between lower extremity muscle strength an speed[7,8] . A person needs at least a minimum level of strength to walk at a given speed. Even children with cerebral palsy who have mild disabilities demonstrate substantial weakness compared with age-related peers[4,6] .

Many treatments exist to improve gait and function of person with cerebral palsy. They include tendon lengthening , transfer or release, selective dorsal rhizotomy , Botulinum toxin, Baclofen, stretching . None of these treatments increase muscle strength.

More than 50 years ago, Phelps contended that resisted exercise to develop strength or skill in a weakened muscle or an impaired muscle group was an integral part of treatment in cerebral palsy[14] .

Directly loading the muscle through specific exercises, activities , or sufficiently intense electrical stimulation is the only direct way to increase muscle strength in cerebral palsy and may be particularly useful in augmenting or maximizing the functional outcomes of other interventions that address different component of the motor disorder[2,3,8].

The basis for producing strength gains in children with cerebral palsy appears to be same as those for people without chronic motor disorders. Children with cerebral palsy also appear to be gain strength at the same rate as persons with weakness that has no CNS pathology in programs of similar intensity and duration[2].

BACKGROUND AND NEED FOR THE STUDY

Children with cerebral palsy walk slowly; they require high energy expenditure, often lack endurance because of muscle weakness. The purpose of this study is to examine whether strengthening lower extremity improves gait speed, cadence, stride length and motor activity in cerebral palsy children.

HYPOTHESIS

Null Hypothesis: There is no significant change in the gait parameters and muscle strength after lower extremity strength training in cerebral palsy children.

Alternate Hypothesis: There is a significant change in the gait parameters muscle strength after lower extremity strength training in cerebral palsy children.

OPERATIONAL DEFINITIONS

CEREBRAL PALSY – Dr. John Little stated that cerebral palsy is a persistent disorder of movement and posture appearing early in life and due to a developmental non progressive disorder of brain.

GAIT is the manner of moving the body from one place to another by alternatively and repetitively changing the location of the feet, with the condition that at least one feet is in contact with the walking surface.

CADENCE – Cadence is the number of the steps taken by a person per unit time. Cadence may be measured the number of steps per second or per minute.

STRIDE LENGTH – Stride length is the linear distance between two successive events that are accomplished by the same lower extremity during gait.

SPEED –Speed of gait is the product of stride length and cadence. It is the scalar quantity or simply the magnitude of the vector velocity measured in meters per second or centimeters per second.

LITERATURE REVIEW

1. **RossSA, EngsbergJR(2007),** no relationship exists between spasticity and strength and that increasing muscle strength does not alter spasticity[5.]

2. **Lee JH, Sung IY, Yoo ZY (2007),** strengthening exercise could be a useful method to improve gait function of children with spastic cerebral palsy. Increased gait speed and stride length & decreased double support phase[31].

3. **Diane L, Damiano (2004),** says that children with cerebral palsy suffer biomechanical misalignment on the one hand and are denied proper use of their muscles on the other.

4. **Joanne Bundonis (2004),** strength training has cardiovascular neuromuscular system benefits as well as psychological**,** fitness and functional benefits[29].

5. **Blunell SW, Shepherd RB, DeanCM, Adams RD, CahillBM (2003),** task specific strengthening exercises run as a group circuit class, resulted in improved strength and functional performance[30].

6. **McBurney et al (2003),** found that the children had increased strength, decreased activity limitations improved mobility after performing a lower extremity strengthening home program[28].

7. **RichSmith Rose (2002),** the idea underlying the energetics of walking is that the amount of energy required to walk, the shorter time and distance an individual will be able to endure ambulation without tiring[32].

8. **Dodd KJ, Taylor DL, Damiano DL (2002),** suggests that training can increase strength and may improve motor activity in people with cerebral palsy without any adverse effects.

9. **Damiano DL, Martellotta TL, Quinlivan J, Abel MF (2001),** in a sample of children with a broader range of involvement, those with greater muscle spasticity in the antagonist tended to have greater agonist weakness.

10. **Teixeira-Salmela LF, Olney SJ, Nadeau S, Brouwer B (1999),** in chronic stroke direct muscle strengthening improved functional performance in persons whose recovery had plateau before this intervention and was not shown to increase spasticity[40].

11. **Damiano DL, Abel MF (1998),** spastic cerebral palsy children reported higher gait velocity primarily as a result of increased cadence, with a greater capacity to walk faster[7].

12. **Damiano DL, Kelly LE, Vaughan CL. (1995),** hamstring strength was measured before and after a quadriceps-strengthening program in children with cerebral palsy to determine whether the program caused an inadvertent increase in strength in the spastic muscle due to abnormal co contraction or stretch responses elicited in the antagonist during agonist strengthening. The quadriceps showed a mean strength increase of more than 50% with no significant change in the hamstring values.

13. **Damiano DL, Kelly LE, Vaughan CL. (1995) Kramer JF, MecPhail HEA. (1994),** leg strength has shown to be related to freely selected walking velocity and to the Gross Motor Function Measure in children and adolescents with cerebral palsy.

14. **Gowland et al (1992),** inadequate requirements of agonist motor neurons and not increased activity in the antagonist is the primary basis for disorders of motor control following a CNS lesion[34].

15. **Kramer, Macphail (1994),** they studied that there was a direct relationship between knee extensor strength and efficient

walking and gross motor ability. They stated that improvements in muscular strength may be associated with improvements in walking efficiency[8].

16. While spasticity was once thought to the primary contributor to the motor dysfunction noted in cerebral palsy, many have challenged this perspective and now consider 'negative' signs such as muscle weakness to be more harmful to function.

17. **Buchner et al (1992),** proposed a theory that there is a curvilinear relationship between gait speed and muscle strength. The benefit of a muscle-strengthening program on gait speed depends of the target group[22].

18. **Mossberg KA, et al (1990),** stated that the children with spastic diplegic cerebral palsy ambulate at about half the speed of the children without of cerebral palsy[25].

19. **Smidt (1990),** defined gait as the manner of moving the body from one place to another by alternatively and repetitively changing the location of feet with condition that at least on foot is contact with walking surface.

Strength training program have shown that an increase in overall strength relates to improved functional changes, these improvements have noted with GMFM measure, increases physical activity & increases self-selected walking velocities.

20. **Sale DG,** neural adaptation to resistance training, Strength increase due to both a neural & muscle hypertrophy components. It has been reported that initial increasing strength are as a result of neural changes and not muscle hypertrophy[6].

21. **Damiano DL, Vaughan CL, Able MF (1995),** muscle weakness is a major impairment in people with cerebral palsy. They eliminated the concern that heavy resistance exercise would elicit unwanted muscle activity in antagonist muscle[2].

22. **Damino, DL Kelly LE, Vaughn CL (1995),** increase in knee strength can improve gait and function in persons with cerebral palsy[3].

23. Children with cerebral palsy not only consume more energy per minute of walking but because they also walk more slowly, their consumption is not very fuel efficient.

24. **Pery et al (1992),** flexion of the hips and knees is called a crouched gait and is often seen in spastic cerebral palsy as a compensatory gait pattern for inadequate hip extension[35, 36].

25. **Rymer & Katz (1989),** there is no agreement for the role of spasticity a positive sign of lesions to neurons of the motor cortex in the loss of functional performance[39].

26. In a single case study, **Horvat (1987),** found increased range of motion in a spastic muscle after strengthening its antagonist, which countered the suspicion of increasing muscle tight-ness resulting from strengthening[27].

27. **Holden and Coworkers (1984, 1986),** used ink patches placed on patient's shoes and a digital stopwatch to derive timing and distance parameters of gait[38].

28. **Dr Bobath (1980),** elaborates that the lesion affects the immature brain and interferes with the maturation of the central nervous system, which has specific consequences in terms of the type of cerebral palsy which develops, its diagnosis, assessment and treatment.

29. **Dr Little club (1959),** described cerebral palsy as a persistent disorder of the movement an posture appearing early in life and due to a developmental non-progressive disorder of the brain.

30. Darcy Ann Umphred Resistance is an important clinical treatment. Resistance is often used to facilitate intrafusal and extrafusal muscle contraction[1].

31. Resistance can be applied manually, mechanically or by the use of gravity in an activity. Resistance also recruits more motor units.

32. **Steindler, (1955)** defined gait as human gait is a constant interplay between loss and recovery of equilibrium and therefore a series of narrowly escaped catastrophes.

33. Time and distance parameters are sensitive measures of gait abnormalities and often so asymmetry, prolonged stance phase, decreased velocity and increased cadence in children with pathology.

34. Speed of gait is a scalar quantity[24].It is simply the magnitude of the vector velocity. Speed of the gait is product of stride length and cadence. It is measured in m/sec or cm/sec.

35. Stride length is the linear distance between two successive events that are accomplished by the same lower extremity during gait.

36. Cadence is the number of steps taken by a person per unit time. cadence may be measured as the number of steps per second or per minute.

37. **Glasscow(1972),**reported on a method of recording an evaluating foot print position on a test walking during a subject's straight walk.

MATERIALS AND METHODOLOGY

3.1 STUDY DESIGN

Experimental design.

3.2 STUDY TYPE

Pretest-posttest randomized control trail.

3.3 STUDY SETTING

The study was one in a clinical setting in cherraan's college of physiotherapy with the consent of parents and children.

3.4 SAMPLE SIZE

No of sample	- 30 subjects.
Experimental group	- 15 subjects.
Control group	- 15 subjects.

3.5 SAMPLING TECHNIQUE

Simple randomized sampling technique.

3.6 STUDY DURATION

The study is done for the period of 5 weeks, 5 days a week,
20-30 minutes per day.

3.7 INCLUSION CRITERIA

- Diplegic cerebral palsy.
- Age 5-14 years children who could walk for at least 45m.
- Children who ambulate independently.
- Children who use assistive devices.
- GMFCS level I & II.

3.8 EXCLUSION CRITERIA

- Subjects who underwent any surgeries within 3 months.
- Subjects who took botox injections within 3 months.
- Children with athetosis, dystonia and other types of cerebral palsy.
- Children with cardio respiratory disease.
- Children with poor mentation.
- Children with severe contractures.

3.9 PROCEDURE

- Intervention: subjects were allocated in 2 groups: control and Experimental group.
- 4 weeks of strength training exercises like bilateral squats, lateral step ups, bilateral heel raises, dorsiflexors strengthening are given for 5 days a week for 5 weeks for the experimental group.
- Control group receives conventional physical therapy.
- Additional resistance would be provided by adding weight to a back pack. An initial training weight would be determined to allow the patient to complete 3 sets of 8 to 10 repetition of each exercise.
- Theraband is used for additional resistance for dorsiflexors.
- I would provide verbal and written instructions in progression of the program.
- In order to monitor an assess progress muscles performance would be measure by MMT and 2 min walk test to obtain a base line measure and would be reassessed every week.

3.10 OUTCOME MEASURES

Subject were evaluated before and after intervention for cadence , step length ,speed, distance walked in 2 minutes, and lower extremity muscle strength.

MATERIALS USED

MATERIALS USED

1. Tray
2. Theraband
3. Trace sheet
4. Cotton
5. Ink
6. Inch tap
7. Backpack with weight
8. Stop clock

DORSIGLEXOR STRENGTHENING

3.10 TESTING OF TOOLS

3.10.1.2-MINUTE WALK TEST

PROCEURE

Children were allowed a minimum of 5 minutes rest before starting the test and were then seated at a starting point inside the outline of a 20m level track. They were told that whenever they were given the instruction to start they were to keep walking around the track as fast possible for 2 minute. They were told that they were not allowed to run. Distance was calculated.

3.10.2 MUSCLE STRENGTH ASSESSMENT

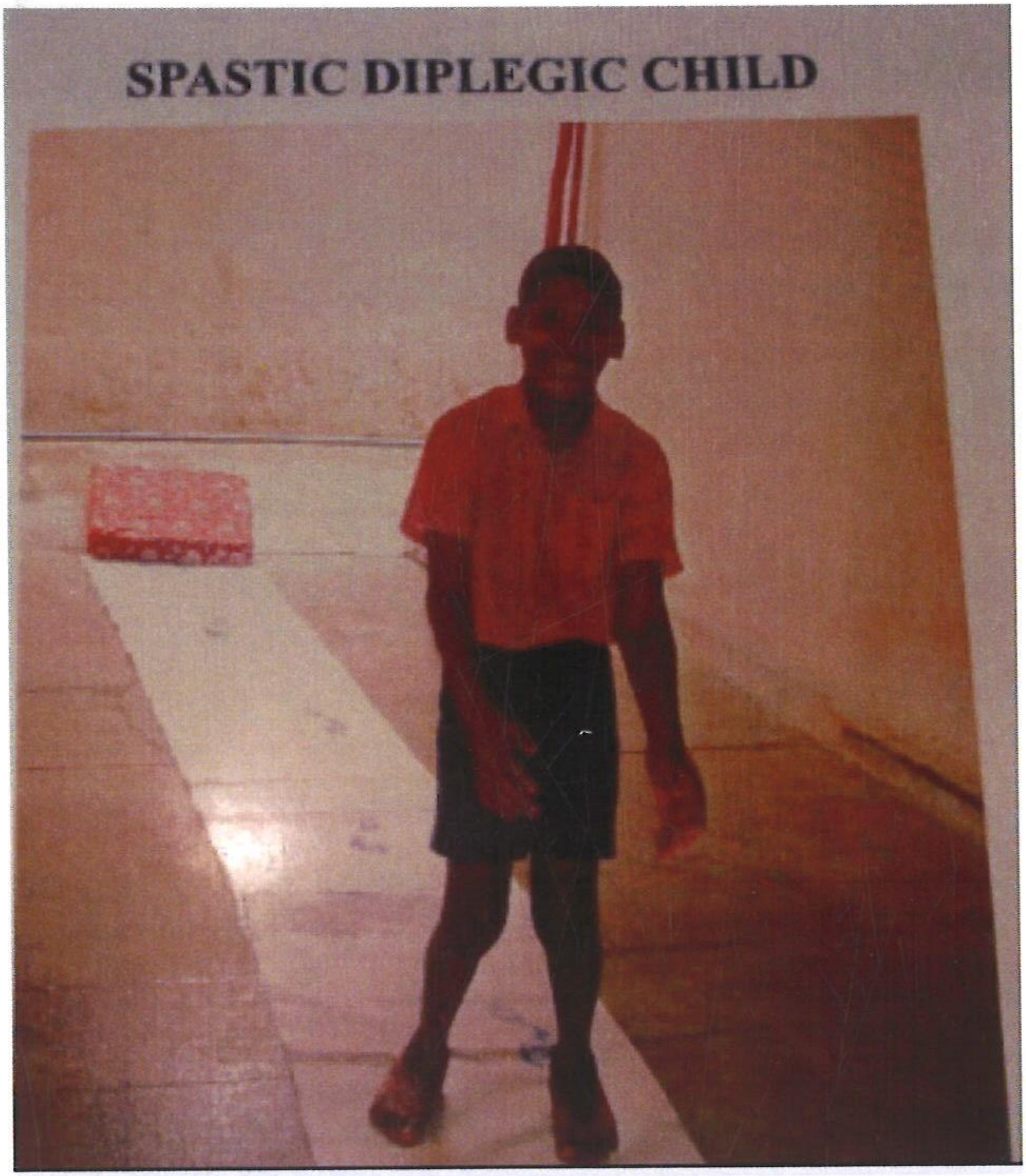
Manual muscle testing is the most common clinical approach to testing strength. **Amundsen, 1990** assesses a subject ability to move a body segment through its ROM or against gravity or against externally applied resistance An ordinal scale is used to grade strength from 0(no contraction) to 5 (full movement against gravity and maximal resistance). The muscle power of hip abductors, hip extensors, knee extensors, dorsiflexors and plantar flexor muscle strength are evaluated in both the group.

3.10.3 GAIT ASSESSMENT

Procedure

The subject is asked to dip his sole of feet in a shallow tray filled with ink and was made to walk on a trace paper. Time taken to walk the distance of 12 feet is noted down. Number of steps and average stride length are noted down from the trace sheets.

GAIT ASSESSMENT



DATA ANALYSIS AND INTERPRETATION

DATA ANALYSIS

The data are analyzed by descriptive and inferential statistics. Mean and standard deviation are calculated by descriptive statistics. Paired t-test is used for within group and independent t-test is used for between groups.

Between group T-Test

Table 1 group statistics

Outcome Measure	Group		Mean	S.D	t- value	Significance Level
Muscle strength	Pre Test	Experimental Group	98	1.4633	1.57263	0.100
		Control Group	91.25	0.72498		
	Post test	Experimental Group	123.75	1.568	4.9798	0.001
		Control Group	100.25	1.9181		

From table1,the muscle strength is statistically significant (0.001) for a t-value of .9798 in the post test.

Table 2 Group Statistics

Outcome Measure	Group		Mean	S.D	t-value	Significance Level
Distance Walked	Pre Test	Experimental Group	63.333	15.69253	0.695	0.497
		Control Group	58.144	15.97209		
	Post test	Experimental Group	78.000	10.01249	2.795	0.013
		Control Group	60.988	15.26618		

From Table2, the parameter distance walked is statically significant (0.013) at the t-value (2.795) between the groups.

Table 3 Group Statistics

Outcome measure	Group		Mean	S.D	t- value	Significance Level
Cadence	Pre Test	Experimental Group	75.61	15.020	0.411	0.687
		Control Group	70.89	31.066		
	Post test	Experimental Group	99.33	27.677	2.189	0.044
		Control Group	77.22	12.337		

From Table 3, the gait parameter cadence is statistically significant (0.044) for a t-value of(2.189) between the two groups.

Table 4 Group statistics

Outcome measure	Group		Mean	S.D	t- value	Significance Level
Speed	Pre Test	Experimental Group	32.444	07.3885	.350	.731
		Control Group	30.850	11.4862		
	Post test	Experimental Group	37.889	06.6086	2.197	.043
		Control Group	30.494	07.6331		

From Table 4, the parameter speed is statistically significant(0.043) for a t-value of (2.189) .

Table 5 Group statistics

Outcome measure	Group		Mean	S.D	t- value	Significance Level
Stride Length	Pre Test	Experimental Group	46.678	08.5041	.453	0.657
		Control Group	45.033	06.8189		
	Post test	Experimental Group	54.756	10.0716	2.676	.017
		Control Group	44.556	05.4130		

From Table 5, the parameter Stride length is statistically significant (0.017) for a t-value of (2.656) between the groups .

Table 6 Experimental Group T Test

Outcome Measure	Group	Mean	S.D	t- value	Significance Level
Distance walked	Pre Test	60.333	15.69235	-3.498	0.008
	Post Test	78.000	10.01249		
Cadence	Pre Test	75.61	15.020	-2.951	0.18
	Post Test	99.33	27.677		
Speed	Pre Test	32.444	07.3885	-2.231	0.05
	Post Test	37.889	06.6086		
Stride Length	Pre Test	46.678	08.5041	-2.537	0.035
	Post Test	54.756	10.0716		
Muscle strength	Pre Test	98	1.7396	2.670411	.025
	Post Test	123.75	1.4632		

From the above table, the mean of distance walked has increased from 66.33(pre test) to 78.00 (post test) and is statistically significant (.008).

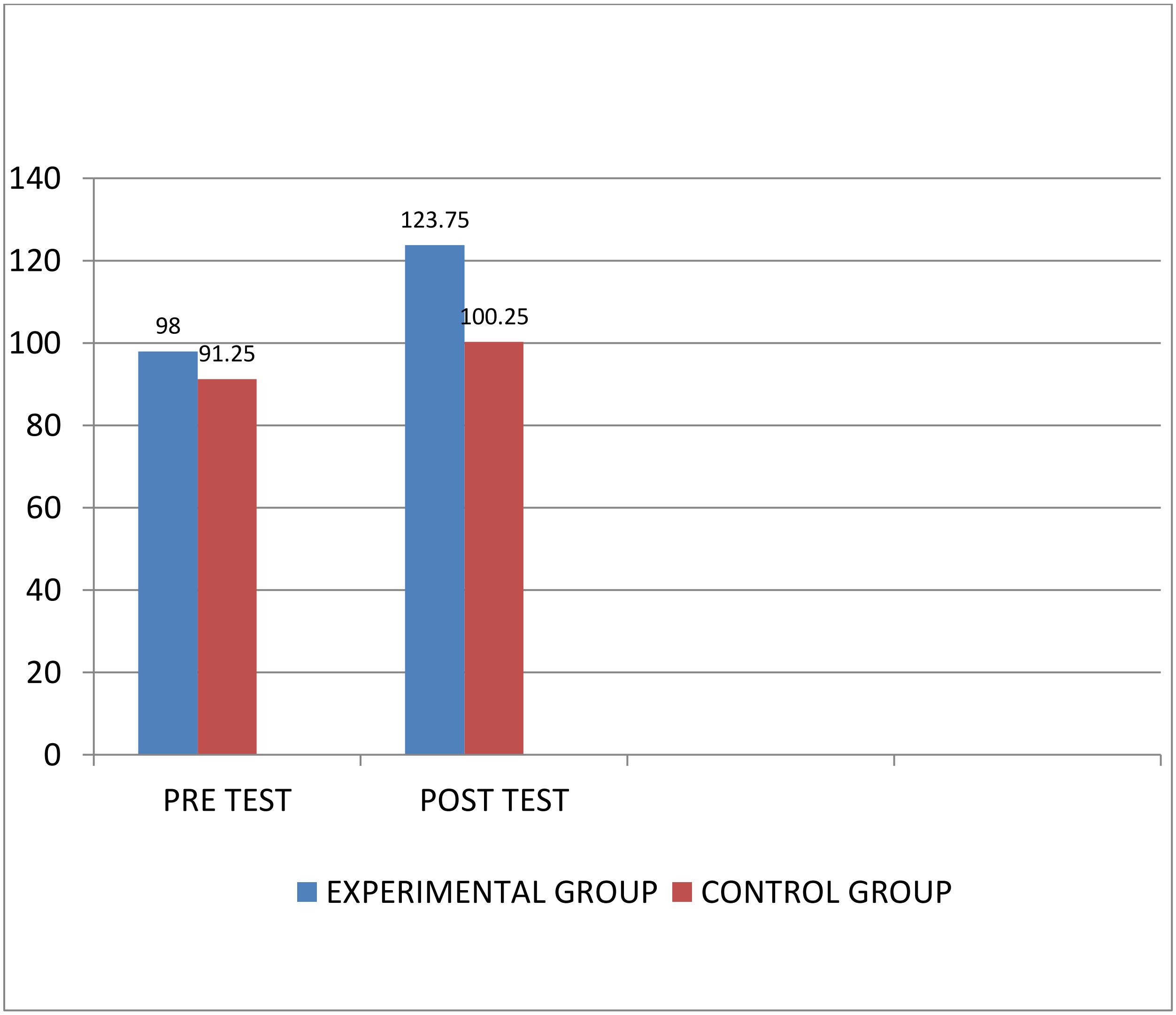
The parameter of gait, cadence has increased from mean value of 75.61 in the pre test to 99.33 in the post test, which is statistically significant(.018).

The mean speed has increased from 32.444(pre test) to 37.889 (post test) which is also statistically significant (.05)

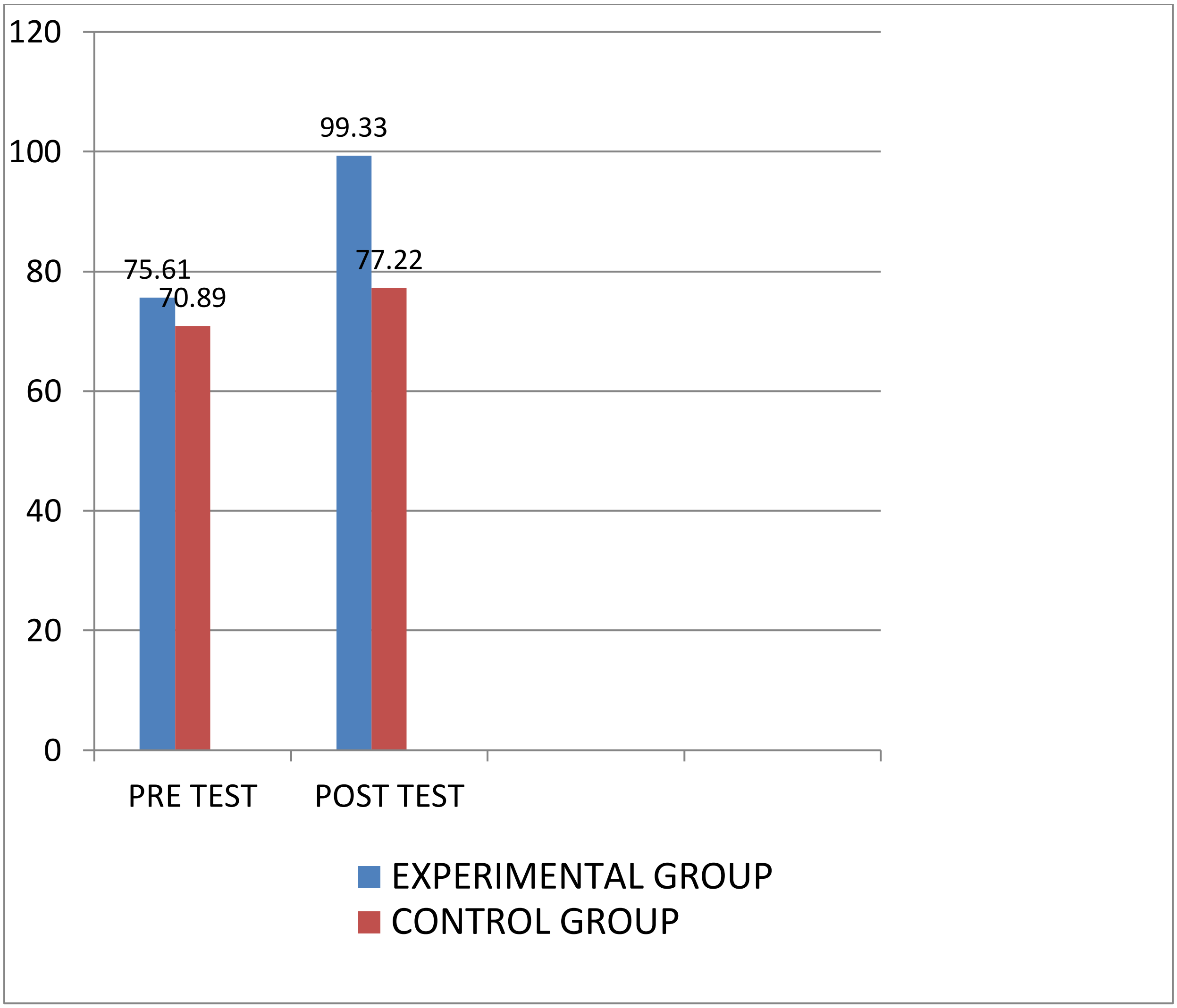
Stride length has increased from its mean value of 46.678 in (pre test) to 54.756, which is also statistically significant (.035)

The muscle strength of the lower extremity muscle has increased from a mean value of 98 in the pre test to 123.75 in the post test which is statistically significant.

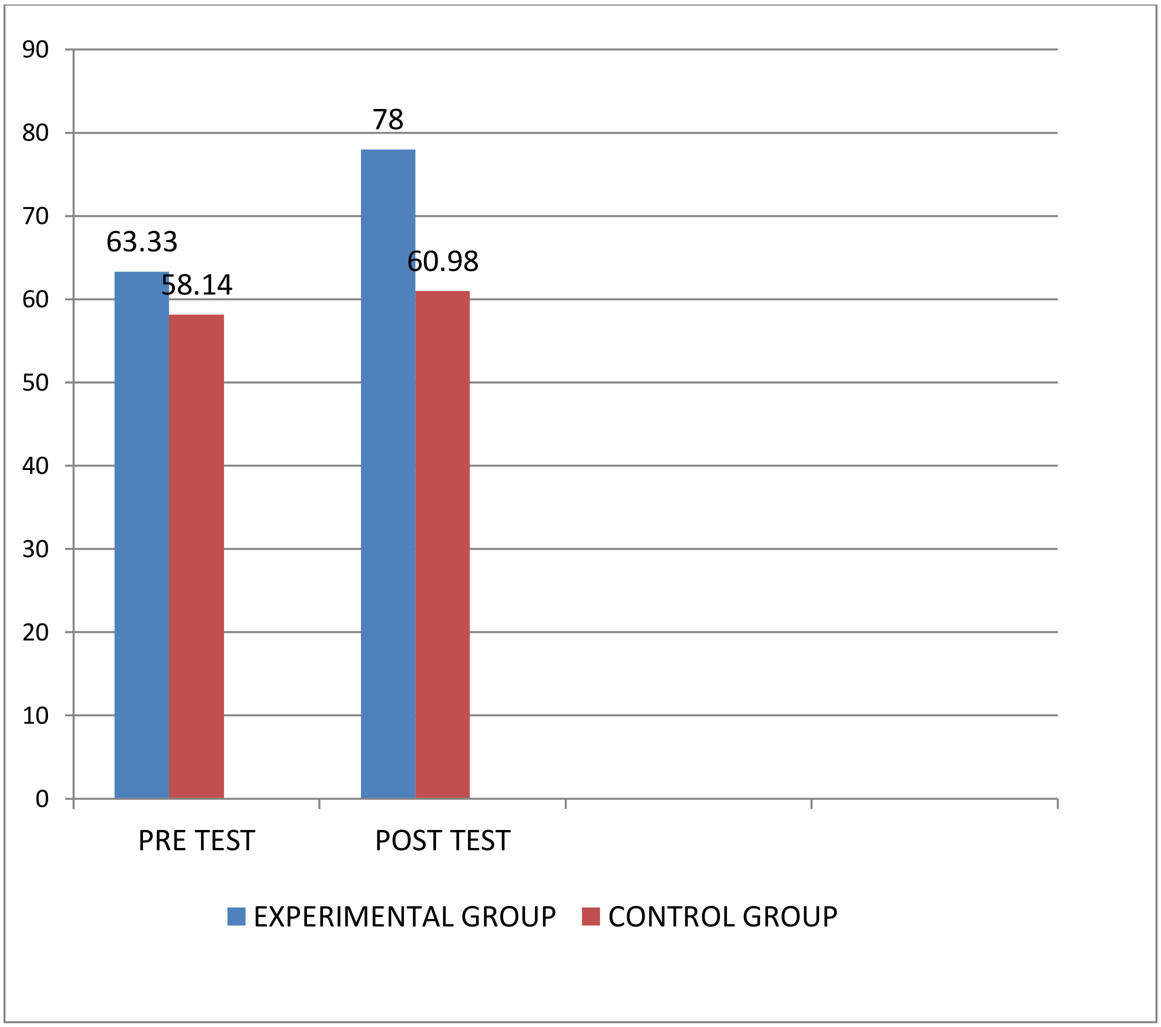
COMPARISON BETWEEN EXPERIMENTAL GROUP AND CONTROL GROUP IN MUSCLE STRENGTH



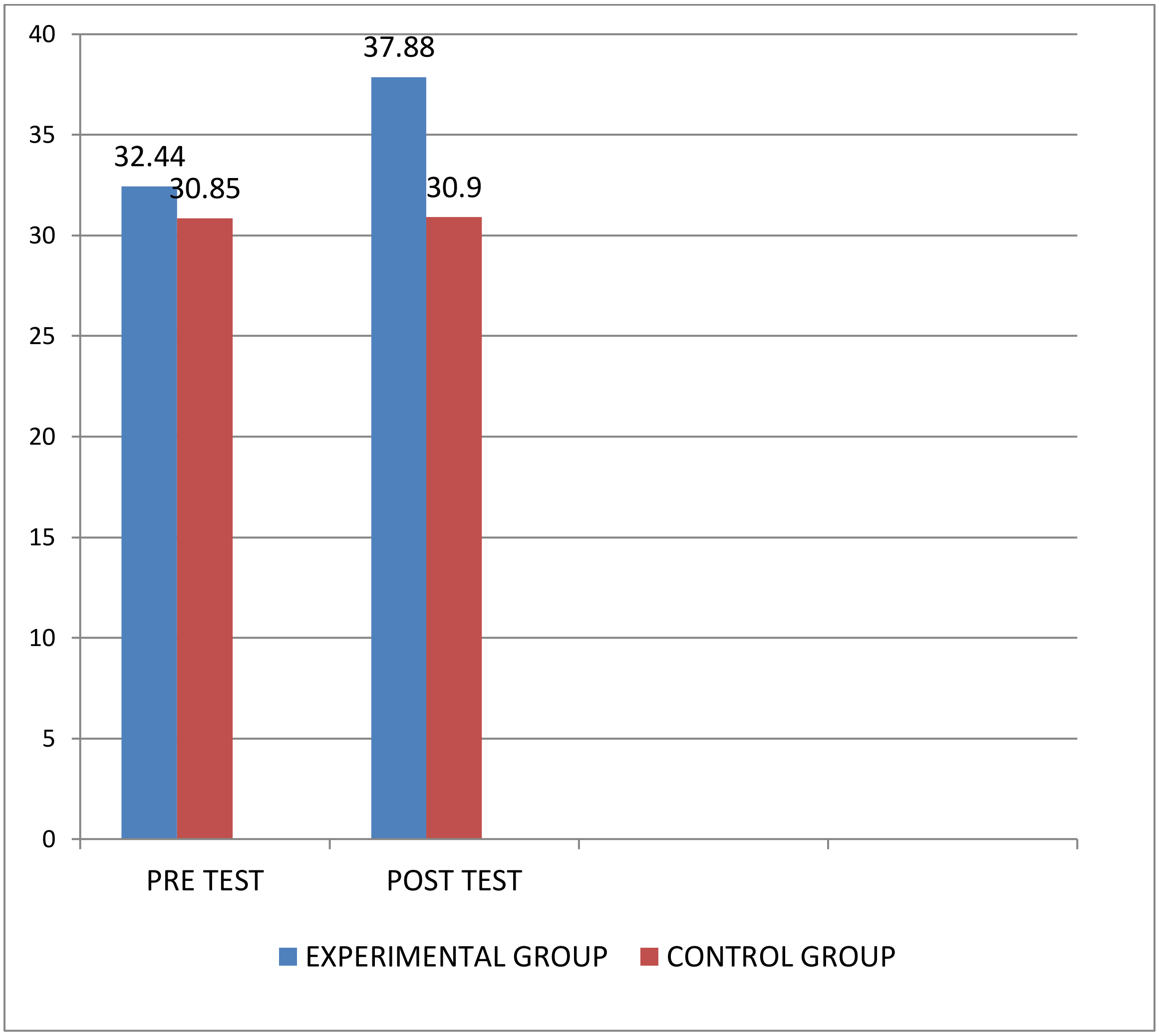
COMPARISON BETWEEN EXPERIMENTAL GROUP AND CONTROL GROUP IN CADENCE



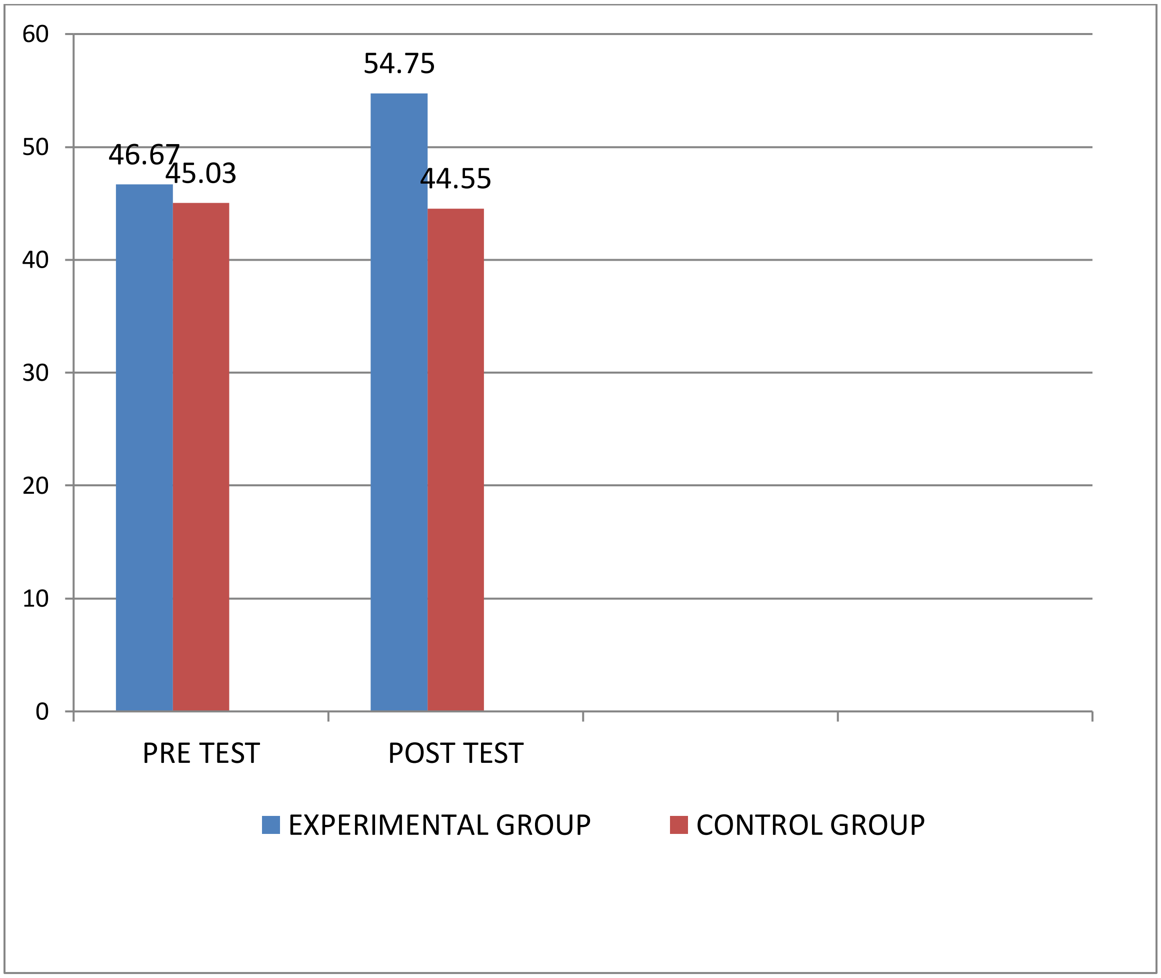
COMPARISON BETWEEN EXPERIMENTAL GROUP AND CONTROL GROUP IN DISTANCE WALK



COMPARISON BETWEEN EXPERIMENTAL GROUP AND CONTROL GROUP IN SPEED



COMPARISON BETWEEN EXPERIMENTAL GROUP AND CONTROL GROUP IN STRIDE LENGTH



DISCUSSION

Cerebral palsy is a disorder of posture and movement that results from a non progressive lesion or injury to immature brain. **Guralnicketal[41]1995** stated that impaired mobility is a critical determinant of independence and a major contributor to physical disability. The type gait abnormality observed depends on the type and extent of CNS pathology, the constellation of resulting impairments.

Crenna and Inverno[24] 1994 have suggested a conceptual framework based on four main impairments contributing to disordered gait in patients with supraspinal lesions.

- Defective muscle activation (Paretic component)
- Abnormal velocity dependent recruitment of muscles during lengthening (spasticity component)
- Loss of selectivity in motor output(co contraction component)
- Changes in mechanical properties of muscle tendon system(non neural component)

Muscles in gait act both concentrically to generate motion eccentrically to control motion thus weakness can result in both inability to generate force to move the body forward an unrestrained motion resulting from lack of muscle control. Muscle strength is more crucial for individuals with disability than for those without. In this study, lower extremity strengthening exercises was done for a period of 5 days a week for 5 weeks.

From table 6, in the experimental group, after intervention the gait parameters improved significantly. The mean distance walked has improved significantly (17.67) after a period of 5 weeks.

The improvement in speed should have primarily occurred through increase in cadence than the stride length.(The mean cadence has increased from 75.61 to 99.33).However stride length has also increased (from 98 in the pre test to 123.75 in the post test)

During the first (2-4) weeks strength can be achieved without structural change in the muscle, but not without neural adaptation. There are more neuro functional changes than structural changes within the muscle.

During the first week of strengthening exercise there is a reduction in the coactivation of other muscles, (muscle synergies, pathological movement) it result in decrease in energy expenditure, movement control improvement . Hence the improved gait performances have occurred because of motor neurological changes.

This improvement is caused by adaptive changes that occur in the nervous system in response to strength training. The EMG findings of high intensity short duration(4 weeks) exercises done in cerebral palsy children have indicated other adaptation mechanisms that may contribute to increased neuronal outflow with training, including increase in maximal firing frequency, increased excitability decreased presynaptic inhibition of spinal motor neurons and down regulation of inhibitory pathways[14].

During the first several weeks of resistive training, gains in strength are almost exclusively neural in nature , meaning the body is leaning to recruit the correct muscles in the proper sequence while inhibiting unnecessary muscle recruitments. The physiologic changes, such as an increase in contractile proteins, stored nutrients, and anaerobic enzymes, take several weeks to develop. **LeMura[15]**states that once the ”learning” phase begins to diminish, remodeling of the muscle is beginning to take place and strength gains continue”.

Short-term resistance training has been reported also to induce hypertrophy of slow and fast muscle fibers, induce alterations in muscle fiber architecture and fiber type distribution and other morphological changes.

From table 7, there were no significant change in the gait parameter after post test in the control group. Although the mean value of distance walked has increased slightly (2.844), it was not statistically significant.

Cadence value increased from a mean value of (70.89 to 77.22), it also it was not statically significant. The other 2 parameters stride length & speed declined slightly from their original mean value.

Although the control group received conventional physical therapy, there were no significant improvements in gait performance within a short interval of 5 weeks.

Mccubbin & shasby[13] reported that repetition alone without the use of resistance did not significantly improve torque production, suggesting that physiological response to muscle loading and not merely motor learning is responsible for increase in muscle strength.

CONCLUSION

All human movements, from the blinking of an eye to the running of a marathon, depends on the proper functioning of skeletal muscle and its strength[39]. A short programme of lower extremity strength training resulted in improved strength and walking efficiency. Hence this randomized control trail suggest that a 5week lower extremity strength training programme is effective in improving muscles strength, cadence, stride length, speed and distance walked in 2 minutes. Hence it can be suggested that the programme of lower extremity strengthening can be incorporated in the therapeutic session to improve the gait performance of cerebral palsy kids.

RECOMMENDATIONS AND LIMITATIONS

RECOMMENDATIONS

- Joint range of motion and spasticity of the agonist muscle may be measured.
- Other groups of cerebral palsy may be trained by strengthening exercises.
- Balance training may also be incorporated along with strength training.
- Large sample size may be used.

LIMITATION

- Small sample size.
- Long term effects are not monitored.
- Other improvements in motor performance are not monitored.

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APPENDICES

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Annexure I

Consent Form

I ___________________ voluntarily agree to allow my child / ward to participate in the study on "the effect of lower extremity strength training on gait in the study on" the effect of children. This study will be carried out for duration of 40 to 45 min, 5 days a week for 5 weeks. All the information given will be kept strictly confidential and used only for research purpose.

Date signature of the parent

Annexure II

Manual muscle testing

MRC grading

Grade 0 - No tension is palpated in the muscle or tendon on maximum voluntary effort

Grade 1 - Tension is palpated in the muscle (or) tendon but no motion occurs.

Grade 2 – Movement with gravity eliminated.

Grade 3 - Movement against gravity .

Grade 4 – Movement against some resistance.

Grade 5 - Normal strength.

Annexure III

GMFCS score sheet

Child's name

Assessment date

GMFCS level

Level I Children walk indoors and outdoors and climb stairs without limitations.

Level II Children walk indoors and outdoors and climb stairs holding onto a railing but experience limitation walking on uneven surfaces and inclines and walking in crowds or confined spaces.

Level III Children walk indoors or outdoors on a level surface with an assistive mobility device and orthoses on level surfaces and climb stairs with support .

Level IV Children may continue to walk for short distances on a walker or rely more on wheeled mobility at home, school and community .

Level V physical impairment restricts voluntary control of movement and the ability to maintain antigravity head and trunk postures. Children have no means of independent mobility and transported.

Annexure IV

ASSESSMENT CHART

Name

Age

Sex

Class

Muscle power assessment

Muscle	Pre test		Post test	
Hip Flexors	**R**	**L**	**R**	**L**
Hip Extensors				
Hip Abductors				
Hip Adductors				
Knee Extensors				
Knee Flexors				
Ankle DF				
Ankle PF				

Gait Function assessment

Name	Age	Speed	Distance walked in two minutes	Cadence	Stride length